

lateral skull x ray film in one patient or by changes in the computed tomogram or air encephalogram in the other three patients. Case 3 required five sequential computed tomograms and an air encephalogram during a period of 5.5 years before the tumour was imaged. In two of our patients (cases 1 and 3) the interval between the onset of symptoms and diagnosis was greater than four years,³ and in one this delay was 11.2 years. This delay occurred in spite of the recent technological advances in neuro-radiological imaging.

These cases illustrate the importance of both endocrine and neuroradiological follow up in children with apparently isolated diabetes insipidus, particularly if the risk of permanent visual loss due to a tumour is to be avoided. While diabetes insipidus may be familial⁴ or associated with other congenital abnormalities of the brain,⁵ the more usual aetiologies are those of intracranial tumour or histiocytosis. The onset of diabetes insipidus in mid-childhood—especially if combined with growth failure—should never be considered idiopathic. Such cases require careful follow up using high resolution computed tomography⁶ and possibly

nuclear magnetic resonance imaging, if necessary into adult life.

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Height in epilepsy

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SUMMARY Longitudinal data on the height of pre-pubertal children with epilepsy are presented. Although both seizures and antiepileptic drugs may affect serum hormone concentrations, linear growth remains normal.

Serum hormone concentrations can be affected by both seizures¹ and antiepileptic drugs.² Suboptimal adult height after somewhat early puberty has been reported in children who attended a residential school because of epilepsy.³ We report on the stature of children with chronic seizure disorders who live at home and attend local schools.

Patients and methods

The medical records of all children with epilepsy who attended the paediatric neurology clinic at our hospital between May and July 1985 were examined. The present data relate to 51 children (29 girls) who

had been assessed when aged 2 to 11 years. Clinical details are given in the Table. Measurements for standing height, using a Harpenden Stadiometer, were available at six monthly intervals. Data from boys and girls were analysed separately.

As calculation of mean trends in mixed longitudinal data, based on mean values at each age, gives inefficient estimates⁴ the age at which the largest numbers of children were measured was selected (8.0 years for girls and 9.5 years for boys) and the mean heights at this age calculated. Individual increments in height were calculated for each six months and the mean increment successively added to or subtracted from the mean height referred to previously.⁵ This method obviates bias effects from single height measurements from unusually tall or short children. More boys and girls were older rather than younger than the reference age. Data were available at 11 years for 14 boys and 10 girls. The parental heights were ascertained in eight cases in whom seizures had been life long and measurements were available at 11 years.

Table Clinical details of the subjects studied

	Boys (n=22)	Girls (n=29)
Predominant seizure type:		
Generalised tonic-clonic	5	5
Myoclonic syndrome	7	7
Absence	1	3
Simple partial	—	2
Complex partial	8	12
Photosensitive	1	—
Antiepileptic drug treatment:		
None	1	—
Barbiturates (including primidone)	7	2
Phenytoin	6	9
Carbamazepine	8	15
Valproate	8	12
Ethosuximide	1	2
Benzodiazepines	8	6
Receiving two drugs	10	14
Receiving three or more drugs	2	2
Age at onset of seizures:		
Before 2 years	13	16
Before 5 years	18	18
Before 7 years	20	23

Results

Figures (a) and (b) show the mean standing heights plotted at six monthly intervals for boys and girls, respectively. The longitudinal curve for boys was between the 25th and 50th centiles for normal boys aged 2 to 11 years. For girls, the growth curve was between the 50th and 75th centiles. There is no evidence of an early onset of pubertal growth in

either sex. The eight children measured at 11 years whose seizures had started in infancy and for whom parental heights were available were growing in keeping with their familial expectations. Subgroups of children with a specific type of seizure, with a high seizure frequency, or receiving particular anti-epileptic drugs had growth curves similar to the total group.

Discussion

Early puberty and suboptimal height attainment has been noted in children with epilepsy in a residential school.³ It has been suggested that the epilepsy in these cases might be particularly severe as only 0.5% of British children with epilepsy attend residential schools. Many of the children in the present study were severely affected and were having frequent daily seizures, but they were living at home and attending day schools. Their growth pattern throughout childhood was within normal limits and puberty did not occur early. It is concluded that seizure frequency does not influence height attainment and that the suboptimal growth of children educated away from home may be related more to concurrent social problems than to epilepsy.

A rise in serum prolactin concentration follows generalised tonic-clonic seizures.¹ Although the rise in growth hormone concentrations is less consistent, there is increased growth hormone responsiveness to stimulation with clonidine after seizures.¹ Despite

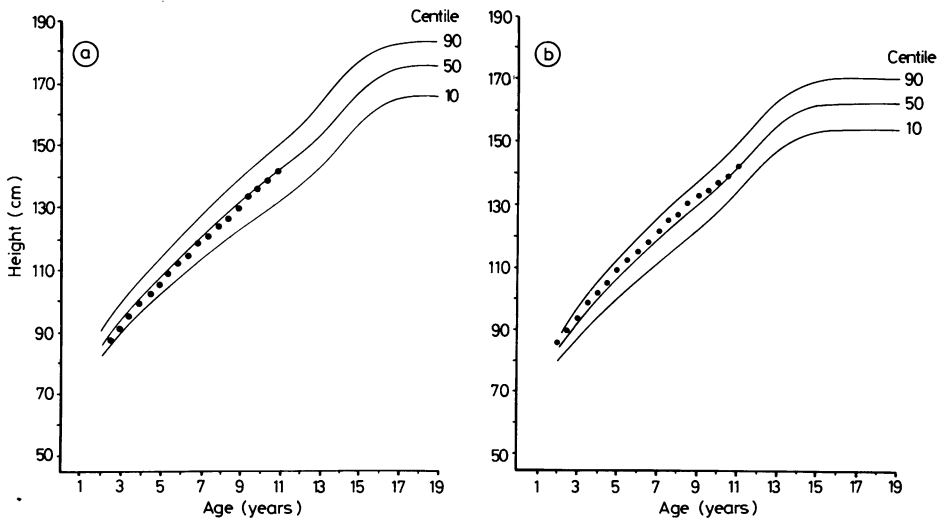


Figure Linear growth in (a) boys and (b) girls with epilepsy. The normal 10th, 50th, and 90th centiles from Tanner height standard charts are shown for comparison.

these reports, we found no evidence that the growth in children with frequent generalised tonic-clonic seizures was impaired relative to those who had infrequent partial seizures.

Phenytoin, phenobarbitone, carbamazepine, and valproate increase the metabolism of sex hormones and enhance the synthesis of sex hormone binding globulin in the liver.² In boys the increase in sex hormone binding globulin leads to an increase in total testosterone concentration, but the concentration of the free fraction remains unaltered.² In adult women increases in serum growth hormone have been noted during treatment with carbamazepine and phenytoin, but the rise in growth hormone caused by insulin hypoglycaemia may be less than expected in adults on antiepileptic drugs.² Comparable data are not available for children. It has been postulated that short stature, which has been reported in adult men with epilepsy,⁶ might be consequent on the use of phenytoin in childhood, but our evidence suggests that antiepileptic drugs do not have clinically important effects on height attainment up to puberty.

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Preoperative stabilisation in congenital diaphragmatic hernia

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SUMMARY Critically ill infants with congenital diaphragmatic hernia were treated by either early surgery or delayed surgery after preoperative stabilisation. The preoperative stabilisation was aimed at correcting acidosis and hypoxia, thereby reducing the severity of persistent fetal circulation. Survival improved from 12.5% after early surgery to 52.9% after delayed surgery.

Infants with congenital diaphragmatic hernia often have severe acidosis and hypoxia at presentation. Immediate repair may lead to an improvement, but many infants deteriorate with increasing hypoxia, dying after a 'honeymoon period' of about 18 hours. This deterioration is usually due to persistent fetal circulation, which is a consequence of hypoxia, acidosis, and the innately abnormal pulmonary arterioles, which are more muscular and reduced in number.^{1,2} Recent reports have suggested that preoperative treatment aimed at correcting acidosis, hypoxia, and tissue hypoperfusion decreases the

severity of persistent fetal circulation and improves survival.³⁻⁵ In 1983 we adopted this new management approach. This paper reports our results.

Patients and methods

From January 1981 to November 1985, 33 infants with congenital diaphragmatic hernia presenting within two hours of birth were treated in our department; their details are shown in Table 1.

Before August 1983, 16 infants were treated with early surgery. Preoperatively most were ventilated only briefly while surgery was organised. Stomach aspiration was used to decompress the bowel. Metabolic acidosis was treated with sodium bicarbonate.

After August 1983, 17 infants were treated with delayed surgery after a period of preoperative stabilisation. Infants in respiratory failure were paralysed with tubocurane and ventilated using low pressures and high rates. Stomach aspiration was again performed. Arterial and venous catheters were inserted through the umbilical vessels. The